cooled blood is returned to the internal organs with beneficial effect. Fans can be used to hasten the process of evaporation.

The patient given the ice treatment in Dallas died the following day.

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Commentary on Male Impotence

TO THE EDITOR: Recently, an article entitled "Impotence Is Not Always Psychogenic" by Spark, White and Connolly appeared in *The Journal of the American Medical Association.*[1] Considerable publicity was given this report in local newspapers (and, I presume, in the lay press nationally). Some of the implications that a lay or professional reader might glean from a cursory glance at the article impel me to write a brief constructive criticism, especially in view of some imperious demands for and assessment of serum testosterone level (and even some clinically questionable need for skull x-ray studies in search for a pituitary tumor) that have come to my attention since the above mentioned study was published.

As a result of my interest during the past 25 years in male psychogenic impotency, I have reported a technique of "urologic" counseling as treatment. To date, more than 300 men with impotency have been evaluated by me on a one-to-one basis. In our 1975 report of 62 such men, we found that more than 90 percent achieved satisfactory restoration of sexual function generally after three weekly sessions.[2] Our more recent report emphasized that, even in the presence of organic illnesses, including diabetes mellitus, or major vascular disease, and of operations such as radical prostatectomy, urologic counseling could aid in reinstatement of sexual function.[3] Thus far I have not assembled my data as to the exact percentage of successful recovery of sexual potency after urologic counseling.

In 1959 my colleagues and I defined sexual potency as "psychologic desire for coitus which produces penile erection adequate for intromission and climax."[4] Frequency of intercourse was specifically excluded from that definition. In the current paper by Spark and co-workers, who quoted Masters and Johnson's definition of impotence as greater than 25 percent erectile failures during attempted intercourse, six of seven men in their hypergonadotrophic-hypogonadism group were said to have engaged in successful intercourse "relatively infrequently" and ". . . episodes of failure caused major embarrassment." For purposes of uniformity, investigators should agree upon a definition and arrange case groupings without variations. Interjection of emotional factors ("embarrassment") clouds the issue. In addition, there is fundamental objection to combining *primary impotency* (never having had sexual experience) and *secondary impotency* in a single group.

Spark and his colleagues commendably reported that determining the serum testosterone level is a valuable step in the study of an impotent patient for the purpose of diagnosing ". . . a surprisingly high incidence of abnormalities of the hypothalamic-pituitary-gonadal axis." However, the conclusion that serum testosterone level may be diagnostically definitive must be challenged since it is well known that men with either normal or low serum testosterone level may be potent or impotent. Indeed a low testosterone level in an impotent man raises the question as to whether it precipitated the impotency or, on the contrary, was a consequence of sexual inactivity. Of the 105 patients in Spark's report, 68 had normal serum testosterone levels.

These authors found low testosterone levels in 37 men never previously evaluated by hormone studies. Among these patients many had significant endocrinopathies: 10 of 20 in the hypogonadotrophic-hypogonadism group had pituitary tumor. Physiologic effect of and subjective impact upon a man with pituitary tumor could well impair libido and potency per se, as would also apply to two other of their patients who had brain tumors and another who suffered pure red blood cell aplasia.

The presence of small or soft testes mentioned by Spark and his colleagues does not necessarily have clinical significance. Furthermore, castration has been performed in countless men with advanced prostatic cancer as a therapeutic step—and many of these men continued to perform sexually despite the debilitating effect of prostatic cancer.

With regard to diabetes mellitus, this writer is far more impressed with those 50 percent to 60

percent of diabetic men who retain their potency than in those allegedly impotent because of their disease. It is well known that potency may be retained, or impotency may develop in a diabetic man without correlation with the severity or the control of the disease. Testosterone therapy alone, or with chorionic gonadotrophin, has not been successful in overcoming impotency in diabetes, according to Kolodny et al.[5]

None of Spark's patients received any placebo. A double-blind study would be very important, particularly since I have learned from my clinical experience that many instances of transitory response (if any at all) to "potency medication" result from a three-way interaction, namely, the quests of an *expectant patient* for help from a purportedly *knowledgeable physician* who gives an *allegedly useful medication*—rather than from any pharmacodynamic effect of the medicine itself.

In a lengthy review of the relationship of hormones to aging, Davis concluded that ". . . loss of sexual potency is so complex a process that it is not justified to attribute it primarily to decreased androgen production."[6] I agree.

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Triamcinolone and Keloids

TO THE EDITOR: The epitome of progress "Triamcinolone for Hypertrophic Scars and Keloids," which appeared in the July 1980 issue, merits comments.

Like Dr. Brody, I believe that intralesional injection of triamcinolone is the best currently available treatment for keloids. However, there are three, not two, commercially available respiratory triamcinolone injection materials. Triamcinolone hexacetonide was not mentioned. This salt is the least soluble, and therefore the longest acting of the respiratory triamcinolone preparations on the market. Intralesionally given corticoids are widely used in dermatologic practice. Like most dermatologists, I prefer triamcinolone acetonide (Kenalog). However, I am not aware of any studies comparing the efficacy of the acetonide with the hexacetonide salts in the treatment of keloids.

Triamcinolone acetonide persists in tissues—and therefore has a period of action of three to four months; not the "up to four or five weeks" claimed by Dr. Brody. This is of practical significance; the maximum keloid regression will usually be observed toward the end of this period, and therefore patients should be seen at three- to four-month intervals.

Dr. Brody suggests that the injections may be either preceded by local infiltration of lidocaine or else the triamcinolone suspension may be mixed with local anesthetic before administration. The latter alternative is poor advice. Triamcinolone acetonide—unlike lidocaine—does not by itself sting on injection. The discomfort of injecting keloids without anesthesia is due to a combination of needle prick pain, plus the pain of injecting fluid under pressure. These are instant, but brief, pains; adding lidocaine to the suspension only adds the lidocaine "sting." If a 30-gauge needle is used, injection into small keloids can usually be done without anesthesia. With large keloids, or when the patient is sensitive, Dr. Brody's first suggestion of preliminary infiltration with lidocaine is the appropriate procedure.

Dr. Brody mentions side effects without clearly distinguishing between local and systemic ones. I would disagree with the arbitrary statement that "The maximum dosage is 60 mg. (1.5 ml) every 30 days." This amount of triamcinolone acetonide once a month will completely suppress endogenous corticoid production, and also cause significant systemic effects. Atrophy is the main local side effect—it is of course recognized that atrophy of the *keloid* is the aim of treatment. Too concentrated a corticoid, or too superficial an injection, will lead to *epidermal* atrophy which can cause a very unsatisfactory result. Epidermal atrophy is more likely to occur with the 40 mg per ml triamcinolone acetonide formulation (Kenalog-40), and that is why I usually start with the 10 mg per ml injection.

A few words about the technique of injection. A fine needle (30-gauge) on a tuberculin syringe equipped with a Luer lock should be used. It takes pressure to get the material into a keloid. The in-